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A Method for Managing Evidential Reasoning in a Hierarchical Hypothesis Space*

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Abstract. Although informal models of evidential reasoning have been successfully applied in automated reasoning systems, it is generally difficult to define the range of their applicability. In addition, they have not provided a basis for consistent management of evidence bearing on hypotheses that are related hierarchically. The Dempster–Shafer (D-S) theory of evidence is appealing because it does suggest a coherent approach for dealing with such relationships. However, the theory's complexity and potential for computational inefficiency have tended to discourage its use in reasoning systems. In this paper we describe the central elements of the D-S theory, basing our exposition on simple examples drawn from the field of medicine. We then demonstrate the relevance of the D-S theory to a familiar expert-system domain, namely the bacterial-organism identification problem that lies at the heart of the MYCIN system. Finally, we present a new adaptation of the D-S approach that achieves computational efficiency while permitting the management of evidential reasoning within an abstraction hierarchy.

1 Introduction

The representation and manipulation of incomplete and imperfect knowledge are issues central to the design of reasoning systems. Drawbacks in traditional probabilistic approaches to the management of such uncertainty led us to develop the *certainty factor* (CF) model of inexact reasoning [15]. The initial CF model was implemented in the medical advice program known as MYCIN and subsequently adapted for use in similar (EMYCIN) systems [3]. However, despite the model's good performance in many task domains, its restrictive assumptions [1] and its inability to deal consistently with hierarchical relationships among values of parameters have left us dissatisfied with

the generality of the approach. We have accordingly been attracted to the mathematical theory of evidence developed by Arthur Dempster. Although it also makes assumptions that do not hold in all problem-solving domains, its coherent approach to the management of uncertainty among hierarchically related hypotheses merits careful study and interpretation in the context of automated reasoning systems.

This theory was first set forth by Dempster in the 1960s and subsequently extended by Glenn Shafer when he published *A Mathematical Theory of Evidence* [14]. The theory's relevance to the issues addressed in the CF model was not immediately recognized [19], but recently researchers have begun to investigate applications of the theory to artificial intelligence systems [2, 6, 7, 10, 11, 16].

An advantage of the Dempster–Shafer (D-S) theory over previous approaches is its ability to model the narrowing of the hypothesis set with accumulation of evidence, a process which characterizes diagnostic reasoning in medicine and expert reasoning in general. An expert uses evidence which may apply not only to single hypotheses but also to sets of hypotheses that together comprise a concept of interest. The functions and combining rule of the D-S theory are well suited to represent this type of evidence and its aggregation.

We believe there are several reasons why the D-S theory is not yet well appreciated by the artificial intelligence research community. One problem has been the mathematical notation used in most of the books and papers that discuss it. In addition, the discussions generally lack simple examples that could add clarity to the theory's underlying notions. Finally, the D-S theory is widely assumed to be impractical for computer-based implementation due to an evidence-combination scheme that assures computational complexity with exponential-time requirements. Although we could not totally avoid mathematical notation in this paper, we do address all three of the issues cited here, paying particular attention to methods for applying the theory in ways that are computationally tractable.

In 1981, Barnett showed that apparent exponential-time requirements of the D-S model could be reduced to simple polynomial time if the theory were applied to single hypotheses, and to their negations, and if evidence were combined in an orderly fashion [2]. However, Barnett's proposal did not solve the larger problem of how to allow evidential reasoning about *sets of hypotheses* in a way that is computationally tractable for complex domains.

In this paper we propose a technique that permits adapting the D-S theory so that hierarchical relationships among hypotheses are handled in a consistent manner. The method builds on Barnett's approach, augmenting it to provide the additional features in a computationally efficient manner. We shall show that the technique requires an assumption (that the hypothesis space can be reduced to a strict hierarchy) and an approximation (it assigns disconfirmatory evidence only to hypotheses with 'meaning' in the domain), but it does manage to capture the major strengths of the D-S theory while achieving a

computationally tractable execution time and, hence, a practical method for its implementation.

We accordingly have three goals in this paper. First, in Sect. 2 we wish to describe for an AI audience the central elements of the D-S theory, avoiding excessive mathematical notation and basing our exposition on simple examples drawn from the field of medicine. In Sect. 3 we demonstrate the relevance of the D-S theory to a familiar expert-system domain, namely the bacterial-organism identification problem that lies at the heart of MYCIN [3]. Since MYCIN's identification rules deal with single hypotheses and ignore hierarchical relationships, the Barnett technique is directly relevant to the program's task. In Sect. 4 we present an adaptation of the D-S approach that allows computationally efficient reasoning within abstraction hierarchies.

The importance of hierarchical relationships among hypotheses can best be appreciated in the setting of a simple example. Consider MYCIN's task of bacterial-organism identification. Here the hypothesis set is a group of over 100 organisms known to the program. By focusing on single organisms (hypotheses), MYCIN's rules and CF model are unable to deal with *groups of organisms* as hypotheses that have explicit relationships to the single bacteria about which knowledge is available. Such relationships, if they exist, must be specified in MYCIN using additional rules; they are not reflected automatically in the structure of the hypothesis space for the domain. When searching for the identity of an infecting organism, however, microscopic examination of a smear showing gram-negative (pink-staining) organisms narrows the hypothesis set of the 100 or so possible organisms to a proper subset. This subset can also be thought of as a new hypothesis: *the organism is one of the gram-negative organisms*. However, this piece of evidence gives no information concerning the relative likelihoods of the individual organisms in the subset. Bayesians[1] might assume equal prior probabilities and distribute the weight of this evidence equally among the gram-negative organisms but, as Shafer points out, they would thus fail to distinguish between uncertainty, or lack of knowledge, and equal certainty. Because the D-S approach allows one to attribute belief to subsets, as well as to individual elements of the hypothesis set, we believe that it is similar to the evidence-gathering process observed when human beings reason at varying levels of abstraction.

A second piece of evidence, such as the morphology (shape) of the organism, narrows the original hypothesis set (the 100 or so bacterial organisms) to a different subset. How does the D-S theory pool this new piece of evidence with the first? Each is represented by a belief function, and the two belief functions thus must be merged using a combination rule to yield a new function. Belief functions assign numerical measures of belief to hypotheses based on observed evidence. In a rule-based expert system, for example, each inferential rule would have its own belief function associated with it, a function

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[1] A thorough discussion of Bayesian theory and its application to medical diagnostic reasoning may be found in [17].

that assigns belief to the consequent based on the evidence in the premise. The combination rule proposed by Dempster, like the Bayesian and CF combining functions, is independent of the order in which evidence is gathered. In fact, the D-S combination rule includes the Bayesian and CF functions as special cases.

Another consequence of the generality of the D-S belief functions is avoidance of the Bayesian restriction that commitment of belief to a hypothesis implies commitment of the remaining belief to its negation, i.e., the assumption that belief in H is equivalent to $P(H)$ so that the resulting belief in NOT-H is $1 - P(H)$. The concept that, in many situations, evidence partially in favor of a hypothesis should *not* be construed as evidence partially against the same hypothesis (i.e., in favor of its negation) was one of the desiderata in the development of the CF model [15]. As in that model, the D-S measures of belief assigned to each hypothesis in the original set need not sum to 1 but may sum to a number less than 1; some of the remaining belief can be allotted to sets of hypotheses that comprise higher-level concepts of interest.

Although the D-S theory includes many of the features of the CF model, its derivation is based on set-theoretic notions which allow explicit and consistent handling of subset and superset relationships in a hierarchy of hypotheses. As we shall show, this feature provides a conceptual clarity that is lacking in the CF model. In the next sections, we motivate the exposition of the theory with a medical example and then discuss the relevance of the theory to systems that reason in hierarchically organized hypothesis spaces.

2 Basics of the Dempster-Shafer Theory

2.1 A Simple Example of Medical Reasoning

Suppose a physician is considering a case of cholestatic jaundice, i.e., the development of a yellow hue to a patient's skin (jaundice) due to elevated blood levels of bilirubin (a pigment produced by the liver). This problem is caused by an inability of the liver to excrete bile normally, often due to a disease within the liver itself (intrahepatic cholestasis) or blockage of the bile ducts outside the liver (extrahepatic cholestasis). In a typical case of this type, the diagnostic hypothesis set might well include two types of intrahepatic cholestasis, hepatitis (Hep) and cirrhosis (Cirr), and two types of extrahepatic cholestasis, gallstones (Gall) and pancreatic cancer (Pan). There are actually more than four causes of jaundice, but we have simplified the example here for illustrative purposes. In the D-S theory, this set of four disorders is called a frame of discernment, denoted Θ or {Hep, Cirr, Gall, Pan}. As noted earlier, the hypotheses in Θ are assumed mutually exclusive and exhaustive.

One piece of evidence considered by the physician might lend support to the diagnosis of intrahepatic cholestasis rather than to a single disease, i.e., it might support the two-element subset of Θ, {Hep, Cirr}. Note that this

subset corresponds to the hypothesis which is the disjunction of its elements, viz. the hypothesis HEP-OR-CIRR. Similarly, the hypothesis extrahepatic cholestasis = {Gall, Pan} = GALL-OR-PAN. Evidence confirming intrahepatic cholestasis to some degree will cause the physician to allot belief to the subset {Gall, Pan}.

Subsequently a new piece of evidence might help the physician exclude hepatitis to some degree. Evidence disconfirming HEP (i.e., disconfirming the set {Hep}) is equivalent to evidence confirming the hypothesis NOT-HEP, which corresponds to the hypothesis CIRR-OR-GALL-OR-PAN or the subset {Cirr, Gall, Pan}. Thus, evidence disconfirming {Hep} to some degree will cause the physician to allot belief to this three-element subset. Note, however, that although evidence disconfirming the set {Hep} may be seen as confirming the set {Cirr, Gall, Pan}, it says nothing about how the belief in the three-element subset should be allocated among the singleton hypotheses {Cirr}, {Gall}, and {Pan}.

As illustrated above, any subset of hypotheses in Θ gives rise to a new hypothesis, which is equivalent to the disjunction of the hypotheses in the subset. Each element in Θ corresponds to a one-element subset (called a singleton). By considering all possible subsets of Θ, denoted 2^{Θ}, the set of hypotheses to which belief can be allotted is enlarged. Henceforth, we use the term 'hypothesis' in this enlarged sense to denote any subset of the original hypotheses in Θ. We shall also hereafter use set notation to refer to the corresponding hypothesis, e.g., {Cirr, Hep} refers to the hypothesis HEP-OR-CIRR, {Pan} refers to the hypothesis PAN, etc.

A diagrammatic representation of 2^{Θ} for the cholestasis example is given in Fig. 1. Note that a set of size n has 2^n subsets. (The empty set, $\emptyset$, is one of these subsets, but is not shown in Fig. 1; it corresponds to a hypothesis known to be false since the hypotheses in Θ are exhaustive.)
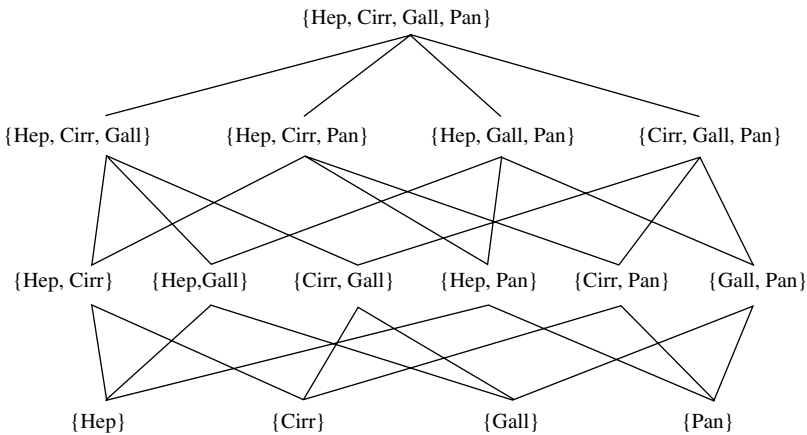

Fig. 1. The subsets of the set of causes of cholestasis

2.2 Basic Probability Assignments

The D-S theory uses a number in the range [0, 1] inclusive to indicate belief in a hypothesis given a piece of evidence. This number is the degree to which the evidence supports the hypothesis.[2] Recall that evidence against a hypothesis is regarded as evidence for the negation of the hypothesis, i.e., for the complement in the set-theoretic interpretation of hypotheses introduced in the previous section. Thus, unlike the CF model, the D-S model avoids the use of negative numbers to represent disconfirming evidence.

The impact of each distinct piece of evidence on the subsets of Θ is represented by a function called a basic probability assignment (*bpa*). A *bpa* is a generalization of a probability mass distribution; the latter assigns a number in the range [0, 1] to every *singleton* of Θ such that the numbers sum to 1. Using 2^{Θ}, the enlarged domain of all subsets of Θ, a *bpa*, denoted m, assigns a number in [0, 1] to every *subset* of Θ such that the numbers sum to 1. (By definition, the number 0 must be assigned to the empty set, since this set corresponds to a false hypothesis.) Thus, m_i allows assignment of a portion of the total belief of 1, based on a given piece of evidence i, to every element in the hierarchy of Fig. 1, not just to those elements on the bottom row as is the case for a probability density function.

The quantity $m(A)$ is a measure of that portion of the total belief committed exactly to A, where A is an element of 2^{Θ}. This portion of belief cannot be further subdivided among the subsets of A and does not include portions of belief committed to subsets of A. Since belief in A certainly entails belief in all subsets of Θ containing A (i.e., nodes 'higher' up in the network of Fig. 1), it would be useful to define a function which computes a *total* amount of belief for each subset in Θ. This function applied to a subset in 2^{Θ}, A, would include not only belief committed exactly to A but to all subsets of A. Such a function, called a belief function in the D-S model, is defined in the next section.

The quantity, $m(\Theta)$, is a measure of that portion of the total belief which is committed to Θ, i.e., which remains unassigned after commitment of belief to various proper subsets of Θ. For example, evidence favoring a single subset A need not say anything about belief in the other subsets. If $m(A) = s$ and m assigns no belief to other subsets of Θ, then $m(\Theta) = 1 - s$. Thus, the remaining belief is assigned to Θ and *not* to the negation of the hypothesis (equivalent to A^c, the set-theoretic complement of A), as would be assumed in the Bayesian model.

Example 1. Suppose there is no evidence concerning the specific diagnosis in a patient with known cholestatic jaundice, i.e., a patient for whom $\Theta = \{\text{Cirr, Hep, Gall, Pan}\}$. The *bpa* representing ignorance, called the vacuous *bpa*, assigns 1 to $\Theta = \{\text{Hep, Cirr, Gall, Pan}\}$ and 0 to every other subset

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[2] Note that this definition corresponds to the notion of a *measure of belief* (MB) in the CF model.

of Θ. Bayesians might attempt to represent ignorance by a function assigning 0.25 to each singleton hypothesis ({Hep}, {Cirr}, {Gall}, and {Pan}), or by a function apportioning the total belief in accordance with information regarding prevalence of the four disorders in the population. As remarked before, however, such functions would imply more information given by the evidence than is truly the case.

Example 2. Suppose that the evidence supports, or confirms, the diagnosis of intrahepatic cholestasis = {Hep, Cirr} to the degree 0.6, but does not support a choice between cirrhosis and hepatitis. The remaining belief, $1 - 0.6 = 0.4$, is assigned to Θ. The hypothesis corresponding to Θ is known to be true under the assumption of exhaustiveness. Thus, $m(\{\text{Hep, Cirr}\}) = 0.6$, $m(\Theta) = m(\{\text{Hep, Cirr, Gall, Pan}\}) = 0.4$ and the value of m for every other subset of Θ is 0. Bayesians might have assigned the remaining belief to extrahepatic cholestasis= {Gall, Pan}, the negation (complement) of intrahepatic cholestasis, rather than to Θ.

Example 3. Suppose that the evidence disconfirms the diagnosis of {Hep} to the degree 0.7. This is equivalent to confirming that of {Cirr, Gall, Pan} to the degree 0.7. Thus, $m(\{\text{Cirr, Gall, Pan}\}) = 0.7$, $m(\Theta) = 0.3$ and the value of m for every other subset of Θ is 0. Note that the notion of disconfirmation does not have a clear correlate in classical probability theory; the CF theory, for example, was developed largely in an effort to address the need to define relationships between confirmation and disconfirmation.

Example 4. Suppose that the evidence confirms the diagnosis of {Hep} to the degree 0.8. Then, $m(\{\text{Hep}\}) = 0.8$, $m(\Theta) = 0.2$, and m is 0 elsewhere.

2.3 Belief Functions

A belief function, denoted *Bel*, corresponding to a specific *bpa, m*, assigns to every subset A of Θ the sum of the beliefs committed exactly to every subset of A by m. For example:

$$\begin{aligned} Bel\left(\{\text{Hep, Cirr, Pan}\}\right) = &\, m\left(\{\text{Hep, Circ, Pan}\}\right) + m\left(\{\text{Hep, Cirr}\}\right) \\ &+ m\left(\{\text{Hep, Pan}\}\right) + m\left(\{\text{Cir, Pan}\}\right) \\ &+ m\left(\{\text{Hep}\}\right) + m\left(\{\text{Cirr}\}\right) + m\left(\{\text{Pan}\}\right). \end{aligned}$$

Thus, $Bel(A)$ is a measure of the *total* amount of belief in A and not the amount committed precisely to A by the evidence corresponding to the *bpa m*.

This relationship may be clarified by referring to Fig. 1. Note that the following observations follow from the definition given:

(1) *Bel* and m are equal for singletons. For example, $Bel(\{\text{Hep}\}) = m(\{\text{Hep}\})$.
(2) $Bel(A)$, where A is any other subset of Θ, is the sum of the values of m for every subset in the subhierarchy formed by using A as root. For example,

$$Bel\,(\text{intrahepatic cholestasis}) = Bel\,(\{\text{Hep, Cirr}\})$$
$$= m\,(\{\text{Hep, Cirr}\}) + m\,(\{\text{Hep}\})$$
$$+ m\,(\{\text{Cirr}\})\,.$$

(3) $Bel(\Theta)$ is always equal to 1 since $Bel(\Theta)$ is the sum of the values of m for every subset of Θ. This sum must be 1 by definition of a *bpa*. Clearly, the total amount of belief in Θ should be equal to the total amount of belief, 1, since the singletons are exhaustive. In Fig. 1, this means that $Bel(\text{cholestatic jaundice}) = Bel(\Theta) = 1$.

To further illustrate, the belief function corresponding to the *bpa* of Example 2 above is given by $Bel(\Theta) = 1$, $Bel(A) = 0.6$, where A is any proper subset of Θ containing $\{\text{Hep, Cirr}\}$, and the value of Bel is 0 for every other subset of Θ.

2.4 Combination of Belief Functions

The evidence-gathering process for diagnosis requires a method for combining the support for a hypothesis, or for its negation, based upon multiple, accumulated observations [15]. The D-S model also recognizes this requirement and provides a formal proposal for its management. Given two *bpa*'s, each with the same frame of discernment Θ but based on two different observations (e.g., two different inferential rules lending positive or negative support to the same or competing hypotheses in an expert system), Dempster's combination rule shown below computes a new *bpa* which represents the impact of the combined evidence.

Concerning the validity of this rule, Shafer writes that although he can provide "no conclusive *a priori* argument,. . .it does seem to reflect the pooling of evidence." In the special case of a frame of discernment containing two elements, Dempster's rule can be found in Johann Heinrich Lambert's book, *Neues Organon*, published in 1764. In another special case where the two *bpa*'s assign evidential support to exactly one and the same hypothesis, the rule reduces to that found in the MYCIN CF model and in *Ars Conjectandi*, the work of the mathematician Jean Bernoulli in 1713. It is based on intuition of how evidence should combine, however, and not on any formal underlying theory.

The Dempster combination rule differs from the CF combining function in the pooling of evidence supporting mutually exclusive hypotheses. For example, evidence supporting $\{\text{Hep}\}$ reduces belief in each of the singleton hypotheses—$\{\text{Cirr}\}$, $\{\text{Gall}\}$, $\{\text{Pan}\}$—and in any disjunction (subset of Θ) not containing $\{\text{Hep}\}$, e.g., $\{\text{Cirr, Gall, Pan}\}$, $\{\text{Cirr, Pan}\}$, etc. As we discuss later, if the D-S model were adapted for use in an EMYCIN system, each new piece

of evidence would have an indirect impact on competing hypotheses, a feature not provided by the CF model. The Dempster combination rule also differs from the CF model in its approach to the assignment of belief in a hypothesis when confirming and disconfirming evidence is pooled.

Let Bel_1, Bel_2 and m_1, m_2 denote two belief functions and their corresponding bpa's, respectively. The D-S combination rule defines a new bpa, denoted $m_1 \oplus m_2$, which represents the combined effect of m_1 and m_2. The corresponding belief function, denoted $Bel_1 \oplus Bel_2$, may then be computed from $m_1 \oplus m_2$ by definition of a belief function.

The Dempster combining function, also known as Dempster's rule, suggests that $m_1 \oplus m_2$ may be calculated from m_1 and m_2 by considering all products of the form $m_1(X)m_2(Y)$ where X and Y are individually varied over all subsets of Θ. It can be shown that the resulting function is itself a bpa since the result of summing all such products is 1 by elementary algebra and the definition of a bpa:

$$\sum m_1(X) m_2(Y) = \sum m_1(X) \sum m_2(Y) = 1 \times 1 = 1.$$

Dempster's rule states that the bpa representing the combination of m_1 and m_2 apportions the total amount of belief among the subsets of Θ by assigning $m_1(X)m_2(Y)$ to the set intersection of X and Y. Note that there are typically several different subsets of Θ whose intersection yields the same subset of Θ. In the cholestatic jaundice example of Fig. 1, for example, the set {Hep, Cirr} will be obtained by intersecting {Hep, Cirr} with any superset of {Hep, Cirr}, by intersecting {Hep, Cirr, Pan} with {Hep, Cirr, Gall}, etc. Thus, for every subset A of Θ, Dempster's rule defines $m_1 \oplus m_2(A)$ to be the sum of all products of the form $m_1(X)m_2(Y)$ where X and Y are selected from the subsets of Θ in all possible ways such that their intersection is A. The commutativity of multiplication ensures that the rule yields the same value regardless of the order in which the functions are combined. This is an important property since evidence aggregation should be independent of the order of its gathering. The following two examples illustrate the combination rule.

Example 5. As in Examples 2 and 3, suppose that for a given patient, one observation supports intrahepatic cholestatic = {Hep, Cirr} to degree 0.6 (m_1) whereas another disconfirms hepatitis (i.e., confirms {Cirr, Gall, Pan}) to degree 0.7 (m_2). Then our net belief based on both observations is given by $m_1 \oplus m_2$. For illustrative purposes, an 'intersection tableau' with values assigned by m_1 and m_2 along the rows and columns, respectively, is a helpful device. Only nonzero values assigned by m_1 and m_2 need be considered since if $m_1(X)$ and/or $m_2(Y)$ is 0, then the product $m_1(X)m_2(Y)$ contributes 0 to $m_1 \oplus m_2(A)$, where A is the intersection of X and Y. Entry i, j in the tableau is the intersection of the subsets in row i and column j. Clearly, a given subset of Θ may occur in more than one location of the tableau. The product of the bpa values is shown below in parentheses next to the subset. The value of

$m_1 \oplus m_2(A)$ is computed by summing the products in the tableau that are noted in parentheses adjacent to each occurrence of A.

m_2 m_1	{Cirr, Gall, Pan} (0.7)	Θ(0.3)
{Hep, Cirr} (0.6)	{Cirr} (0.42)	{Hep, Cirr} (0.18)
Θ (0.4)	{Cirr, Gall, Pan} (0.28)	Θ (0.12)

In this example, each subset appears only once in the tableau and $m_1 \oplus m_2$ is easily computed:

$$m_1 \oplus m_2 \left(\{\text{Cirr}\}\right) = 0.42,$$
$$m_1 \oplus m_2 \left(\{\text{Hep, Cirr}\}\right) = 0.18,$$
$$m_1 \oplus m_2 \left(\{\text{Cirr, Gall, Pan}\}\right) = 0.28,$$
$$m_1 \oplus m_2 \left(\Theta\right) = 0.12,$$
$$m_1 \oplus m_2 \text{ is 0 for all other subsets of } \Theta.$$

Since $Bel_1 \oplus Bel_2$ is fairly complex, we give only a few sample values:

$$Bel_1 \oplus Bel_2 \left(\{\text{Hep, Cirr}\}\right) = m_1 \oplus m_2 \left(\{\text{Hep, Cirr}\}\right) + m_1 \oplus m_2 \left(\{\text{Hep}\}\right)$$
$$+ m_1 \oplus m_2 \left(\{\text{Cirr}\}\right)$$
$$= 0.18 + 0 + 0.42$$
$$= 0.60,$$
$$Bel_1 \oplus Bel_2 \left(\{\text{Cirr, Gall, Pan}\}\right) = m_1 \oplus m_2 \left(\{\text{Cirr, Gall, Pan}\}\right)$$
$$+ m_1 \oplus m_2 \left(\{\text{Cirr, Gall}\}\right)$$
$$+ m_1 \oplus m_2 \left(\{\text{Cirr, Pan}\}\right)$$
$$+ m_1 \oplus m_2 \left(\{\text{Gall, Pan}\}\right)$$
$$+ m_1 \oplus m_2 \left(\{\text{Cirr}\}\right)$$
$$+ m_1 \oplus m_2 \left(\{\text{Gall}\}\right) + m_1 \oplus m_2 \left(\{\text{Pan}\}\right)$$
$$= 0.28 + 0 + 0 + 0 + 0.42 + 0 + 0$$
$$= 0.70,$$
$$Bel_1 \oplus Bel_2 \left(\{\text{Hep, Cirr, Pan}\}\right) = Bel_1 \oplus Bel_2 \left(\{\text{Hep, Cirr}\}\right) = 0.60,$$

since

$$m_1 \oplus m_2 \left(\{\text{Hep, Cirr, Pan}\}\right) = m_1 \oplus m_2 \left(\{\text{Hep, Pan}\}\right)$$
$$= m_1 \oplus m_2 \left(\{\text{Cirr, Pan}\}\right) = 0.$$

In this example, the reader should note that $m_1 \oplus m_2$ satisfies the definition of a *bpa*: $\Sigma m_1 \oplus m_2(X) = 1$ where X varies over all subsets of Θ, and $m_1 \oplus m_2(\emptyset) = 0$. We have already shown that the first condition in the definition of a *bpa* is always fulfilled, i.e., the sum of the beliefs assigned to all subsets in Θ by the Dempster rule will always sum to 1. However, the second condition (viz. that a *bpa* assign 0 to the empty set) is problematic in cases where the 'intersection tableau' contains $\emptyset$. This situation did not occur in Example 5 because every two sets with nonzero *bpa* values always had at least one element in common. In general, nonzero products of the form $m_1(X)m_2(Y)$ will be assigned to $\emptyset$ whenever X and Y have nonzero *bpa* values but their intersection is the empty set.

The D-S model deals with this problem by setting $m_1 \oplus m_2(\emptyset)$ equal to 0 and normalizing the remaining *bpa* assignments so that they continue to sum to 1.[3] This behavior is achieved by defining κ as the sum of all nonzero values assigned to $\emptyset$ in a given case ($\kappa = 0$ in Example 5). Dempster then divides all other values of $m_1 \oplus m_2$ by $1 - \kappa$. The revised values still sum to 1 and hence satisfy that condition in the definition of a *bpa*. This approach is illustrated by the following example.

Example 6. Suppose now that, for the same patient as in Example 5, a third belief function (m_3) corresponds to a new observation which confirms the diagnosis of hepatitis to the degree 0.8 (i.e., suppose we have a combination of Examples 4 and 5). We now need to compute $m_3 \oplus m_4$, where $m_4 = m_1 \oplus m_2$ of Example 5.

m_3 \ m_4	{Cirr} (0.42)	{Hep, Cirr} (0.18)	{Cirr, Gall, Pan} (0.28)	Θ (0.12)
{Hep} (0.8)	$\emptyset$ (0.336)	{Hep} (0.144)	$\emptyset$ (0.224)	{Hep} (0.096)
Θ (0.2)	{Cirr} (0.084)	{Hep, Cirr} (0.036)	{Cirr, Gall, Pan} (0.056)	Θ (0.024)

In this example, there are two null entries in the tableau, one assigned the value 0.336 and the other 0.224. Thus:

$$\kappa = 0.336 + 0.224 = 0.56 \quad \text{and} \quad 1 - \kappa = 0.44,$$
$$m_3 \oplus m_4(\{\text{Hep}\}) = (0.144 + 0.096)/0.44 = 0.545,$$
$$m_3 \oplus m_4(\{\text{Cirr}\}) = 0.084/0.44 = 0.191.$$
$$m_3 \oplus m_4(\{\text{Hep, Cirr}\}) = 0.036/0.44 = 0.082,$$
$$m_3 \oplus m_4(\{\text{Cirr, Gall, Pan}\}) = 0.056/0.44 = 0.127,$$
$$m_3 \oplus m_4(\Theta) = 0.024/0.44 = 0.055,$$
$$m_3 \oplus m_4 \text{ is 0 for all other subsets of } \Theta.$$

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[3] This convention is intuitive in that it maintains the relative beliefs among the rest of the hypotheses in 2^Θ. It should be noted, however, that the normalization convention is not supported in any theoretic sense and can lead to paradoxical behavior of the model in certain settings [19]. Some have argued that it would be just as rational to move the belief originally assigned to $\emptyset$ to Θ.

Note that $\sum m_3 \oplus m_4(X) = 1$, as is required by the definition of a *bpa*.

2.5 Belief Intervals

After combining all *bpa*'s with the same frame of discernment and then computing the belief function *Bel* defined by this new *bpa*, how should the information given by *Bel* be used? *Bel(A)* gives the total amount of belief committed to the subset A after all evidence bearing on A has been pooled. However, the function *Bel* contains additional information about A, namely $Bel(A^c)$, the extent to which the evidence supports the negation of A. The quantity $1 - Bel(A^c)$ expresses the plausibility of A, i.e., the maximum extent to which the current evidence could allow one to believe A (note that this is *not* the same as *Bel(A)*, the extent to which the current evidence specifically supports A).

The information contained in *Bel* concerning a given subset A may be conveniently expressed by the interval:

$$[Bel(A), 1 - Bel(A^c)].$$

It is not difficult to see that the left endpoint is always less than or equal to the right: $Bel(A) \leq 1 - Bel(A^c)$, or equivalently, $Bel(A) + Bel(A^c) \leq 1$. Since $Bel(A)$ and $Bel(A^c)$ are the sum of all values of m for subsets of A and A^c, respectively, and since A and A^c have no subsets in common, $Bel(A) + Bel(A^c) \leq \Sigma m(X) = 1$ where X varies over all subsets of Θ.

In the Bayesian situation, in which $Bel(A) + Bel(A^c) = 1$, the two endpoints of the belief interval are equal and the width of the interval, $1 - Bel(A^c) - Bel(A)$, is 0. In the D-S model, however, the width is usually not 0 and is a measure of the belief which, although not committed to A, is also not committed to A^c. It may be seen that the width is the sum of belief committed exactly to subsets of Θ which intersect A but which are not subsets of A. If A is a singleton, all such subsets are supersets of A, but this is not true for a nonsingleton A. To illustrate, let $A = \{\text{Hep}\}$ and refer to Fig. 1:

$$
\begin{aligned}
1 - Bel(A^c) - Bel(A) &= 1 - Bel(\{\text{Cirr, Gall, Pan}\}) - Bel(\{\text{Hep}\}) \\
&= 1 - [m(\{\text{Cirr, Gall, Pan}\}) + m(\{\text{Cirr, Gall}\}) \\
&\quad + m(\{\text{Cirr, Pan}\}) + m(\{\text{Gall, Pan}\}) + m(\{\text{Cirr}\}) \\
&\quad + m(\{\text{Gall}\}) + m(\{Pan\})] - m(\{\text{Hep}\}) \\
&= m(\{\text{Hep, Cirr}\}) + m(\{\text{Hep, Gall}\}) \\
&\quad + m(\{\text{Hep, Pan}\}) + m(\{\text{Hep, Cirr, Gall}\}) \\
&\quad + m(\{\text{Hep, Cirr, Pan}\}) + m(\{\text{Hep, Gall, Pan}\}) \\
&\quad + m(\Theta).
\end{aligned}
$$

Belief committed to a superset of {Hep} might, upon further refinement of evidence, result in belief committed to {Hep}. Thus, the width of the belief

interval is a measure of that portion of the total belief, 1, which could be added to that committed to {Hep} by a physician willing to ignore all but the disconfirming effects of the evidence.

The width of a belief interval can also be regarded as the amount of uncertainty with respect to a hypothesis given the evidence. It is belief which is committed to neither the hypothesis nor the negation of the hypothesis by the evidence. The vacuous belief function results in width 1 for all belief intervals and Bayesian functions result in width 0. Most evidence leads to belief functions with intervals of varying widths where the widths are numbers between 0 and 1.

3 The Dempster-Shafer Theory Applied to Singleton Hypotheses

Despite the intuitive appeal of many aspects of the D-S theory outlined above, the enumeration of all subsets of Θ in the application of the Dempster combining rule becomes computationally intractable when there are a large number of elements in Θ (as is true for many real-world problems in which the evidence-gathering scheme could otherwise be employed). If we restrict the hypotheses of interest in 2^{Θ} to the mutually exclusive singletons and their negations, however, Barnett has shown that a linear-time algorithm will permit rigorous application of the Dempster rule [2]. In this section we show that one expert system, MYCIN, can be viewed as a reasoning program in which the principal hypotheses are restricted to singletons. MYCIN will therefore be discussed to illustrate the applicability of the D-S theory in general and the relevance of the Barnett formulation in particular.

MYCIN's representation may be simply recast in terms of the D-S theory we have outlined. A frame of discernment in MYCIN, for example, is a clinical parameter (attribute) which may take on a range of values. The possible values are mutually exclusive and may therefore be seen as the competing hypotheses that make up the elements in Θ.[4] This condition may be a stumbling block to the model's implementation in systems where mutual exclusivity does not generally hold.

The belief functions which represent evidence in MYCIN correspond to the individual rules in the system's knowledge base. These are of a particularly simple form (the CF in a rule corresponds to the value assigned by a *bpa* to the hypothesis in the rule's conclusion based on the evidence in its premise). These features will now be discussed and illustrated with examples.

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[4] Some parameters in MYCIN can take on multiple values, e.g., the patient's drug allergies [3], but we will be focussing here on the central inferences in the system, such as an organism's identity, which satisfy the mutual exclusivity requirement.

3.1 Frames of Discernment

How should the frames of discernment for a reasoning system be chosen? Shafer points out [14] that:

> It should not be thought that the possibilities that comprise Θ will be determined and meaningful independently of our knowledge. Quite to the contrary: Θ will acquire its meaning from what we know or think we know; the distinctions that it embodies will be embedded within the matrix of our language and its associated conceptual structures and will depend on those structures for whatever accuracy and meaningfulness they possess.

The 'conceptual structures' in MYCIN, for example, are the associative triples found in the conclusions of the rules [3]. These have the form (object, attribute, value), i.e., each triple corresponds to a singleton hypothesis of the form 'the attribute of object is value'. As mentioned previously, a frame of discernment would then consist of all triples with the same object and attribute.

For example, one frame of discernment is generated by the set of all triples of the form (Organism-1, Identity, X), where X ranges over all possible identities of organisms known to MYCIN—Klebsiella, E.coli, Pseudomonas, etc. Another frame is generated by replacing 'Organism-1' with 'Organism-2'. A third frame is the set of all triples of the form (Organism-1, Morphology, X), where X ranges over all known morphologies—coccus, rod, pleomorph, etc.

Although it is true that a patient may be infected by more than one organism, these organisms are represented as separate objects in MYCIN (not as separate values of the same parameter for a single object). Thus MYCIN's representation scheme for the parameter that corresponds to its major classification task (i.e., the identity of an organism) complies with the mutual-exclusivity demand for frames of discernment in the D-S theory. Many other expert systems meet this demand less easily. Consider, for example, how the theory might be applicable in a system which gathers and pools evidence concerning a patient's diagnosis. Then there is often the problem of multiple, coexistent diseases, i.e., the hypotheses in the frame of discernment may not be mutually exclusive. One way to overcome this difficulty is to choose Θ to be the set of all subsets of all possible diseases. The computational implications of this choice are harrowing since if there are 600 possible diseases (the approximate scope of the INTERNIST-1 knowledge base [12]), then $|\Theta| = 2^{600}$ and $|2^{\Theta}| = 2^{2^{600}}$! However, since the evidence may actually focus on a small subset of 2^{Θ}, the computations need not be intractable because the D-S theory need not depend on explicit enumeration of all subsets of 2^{Θ} when many have a belief value of zero. An alternative would be to apply the D-S theory after partitioning the set of diseases into groups of mutually exclusive diseases and considering each group as a separate frame of discernment. The latter approach would be similar to that used in INTERNIST-1 [12], where

scoring and comparison of hypotheses is undertaken only after a partitioning algorithm has separated evoked hypotheses into subsets of mutually exclusive diagnoses.

3.2 Rules as Basic Probability Assignments

In the most general situation, a given piece of evidence supports many of the subsets of Θ, each to varying degrees. However, the simplest situation is that in which the evidence supports or disconfirms only one singleton subset to a certain degree and the remaining belief is assigned to Θ. Because of the modular way in which knowledge is captured and encoded in MYCIN, this latter situation applies in the case of its rules.

If the premises confirm the conclusion of a rule with degree s, then the rule's effect on belief in the subsets of Θ can be represented by *bpa*. This *bpa* would assign s to the singleton corresponding to the hypothesis in the conclusion of the rule, call it A, and $1 - s$ to Θ. In the language of MYCIN, the CF associated with this conclusion is s. Since there is no concept equivalent to Θ in MYCIN, however, the remaining belief, $1 - s$, is left unassigned. If the premise of a rule disconfirms the conclusion with degree s, then the corresponding *bpa* would assign s to the subset corresponding to the negation of the conclusion, A^c, and $1 - s$ to Θ. The CF associated with his conclusion is $-s$. Thus, we are suggesting that the CF's associated with rules in MYCIN, and other EMYCIN systems, can be viewed as *bpa*'s in the D-S sense. Note, however, that MYCIN's rules do not permit inferences regarding nonsingleton hypotheses in 2^{Θ}, e.g., the conclusion that an organism is either an E.coli or a Klebsiella, which corresponds to the two-element subset {E.coli, Klebsiella}. Our suggested solution to this problem is outlined in Sect. 4.

3.3 Dempster's Rule Applied to Singleton Hypotheses

If we continue the analogy between CF's in MYCIN's rules and *bpa*'s in the D-S theory, we can consider the use of Dempster's rule for combining belief when two or more rules succeed and assign belief to the same or competing singleton hypotheses. To illustrate, we consider a frame of discernment Θ consisting of all associative triples of the form (Organism-1, Identity, X) where X ranges over all possible identities of organisms known to MYCIN. The triggering of two rules that affect belief in such triples can be categorized in one of three ways:

(1) they may both confirm or both disconfirm the same hypothesis;
(2) one may confirm and the other may disconfirm the same hypothesis;
(3) each may bring evidence to bear on different competing hypotheses.

We describe the approach to each of these possibilities below.

Theorem 1. *Two rules are both confirming or both disconfirming of the same triple, or conclusion. For example, both rules confirm Pseudomonas (Pseu),*

one to degree 0.4 and the other to degree 0.7. The effect of triggering the rules is represented by bpa's, m_1 and m_2, where $m_1(\{\text{Pseu}\}) = 0.4$, $m_1(\Theta) = 0.6$, and $m_2(\{\text{Pseu}\}) = 0.7$, $m_2(\Theta) = 0.3$. The combined effect on belief is given by $m_1 \oplus m_2$, computed using the tableau below:

m_1 \ m_2	{Pseu} (0.7)	Θ (0.3)
{Pseu} (0.4)	{Pseu} (0.28)	{Pseu} (0.12)
Θ (0.6)	{Pseu} (0.42)	Θ (0.18)

Note that $k = 0$ in this example, so normalization is not required (i.e., $1 - k = 1$).

$$m_1 \oplus m_2\,(\{\text{Pseu}\}) = 0.28 + 0.12 + 0.42 = 0.82,$$
$$m_1 \oplus m_2\,(\Theta) = 0.18.$$

Note that $m_1 \oplus m_2$ is a *bpa* which, like m_1 and m_2, assigns some belief to a certain subset of Θ, {Pseu}, and the remaining belief to Θ. For two confirming rules, the subset is a singleton; for disconfirming rules, the subset is a set of size $n - 1$, where n is the size of Θ.[5]

Theorem 2. *One rule is confirming and the other disconfirming of the same singleton hypothesis. For example, one rule confirms {Pseu} to degree 0.4 and the other disconfirms {Pseu} to degree 0.8. The effect of triggering these two rules is represented by bpa's m_1, m_3 where m_1 is defined in the previous example and $m_3(\{\text{Pseu}\}^c) = 0.8$, $m_3(\Theta) = 0.2$. The combined effect on belief is given by $m_1 \oplus m_3$.*

m_1 \ m_2	$\{\text{Pseu}\}^c$ (0.8)	Θ (0.2)
{Pseu} (0.4)	$\emptyset$ (0.32)	{Pseu} (0.08)
Θ (0.6)	$\{\text{Pseu}\}^c$ (0.48)	Θ (0.12)

This time the tableau does contain the empty set as an entry; therefore $k = 0.32$ and $1 - k = 0.68$.

$$m_1 \oplus m_3\,(\{\text{Pseu}\}) = 0.08/0.68 = 0.118,$$
$$m_1 \oplus m_3\,(\{\text{Pseu}\}^c) = 0.48/0.68 = 0.706,$$
$$m_1 \oplus m_3\,(\Theta) = 0.12/0.68 = 0.176,$$
$$m_1 \oplus m_3 \text{ is 0 for all other subsets of } \Theta.$$

---

[5] Note that in this case Dempster's rule has provided the same result as would the original CF combining function (MYCIN would also combine 0.4 and 0.7 to get 0.82; see [15]).

Given m_1 above, the belief interval of {Pseu} is initially $[Bel_1(\{\text{Pseu}\}),$ $1 - Bel_1(\{\text{Pseu}\}^c)] = [0.4,\ 1]$. After combination with m_3, it becomes $[0.118,$ $0.294]$. Similarly, given m_3 alone, the belief interval of {Pseu} is $[0, 0.2]$. After combination with m_1, it becomes $[0.118, 0.294]$.

As is illustrated in this example, an essential aspect of Dempster's rule is the effect of evidence that supports a hypothesis in 2^Θ in reducing belief in other hypotheses in 2^Θ that are disjoint from the supported hypothesis. Thus, evidence confirming $\{\text{Pseu}\}^c$ will reduce the effect of evidence confirming {Pseu}; in this case the degree of support for {Pseu}, 0.4, is reduced to 0.118. Conversely, evidence confirming {Pseu} will reduce the effect of evidence confirming $\{\text{Pseu}\}^c$; 0.8 is reduced to 0.706. These two effects are reflected in the modification of the belief interval of {Pseu} from $[0.4, 1]$ to $[0.118, 0.294]$, where $0.294 = 1 - Bel(\{\text{Pseu}\}^c) = 1 - 0.706$.

Consider the application of the CF combining function ($\text{CF}_{\text{combine}}$) to this same situation.[6] If CF_p is the positive (confirming) CF for {Pseu}, and CF_n is the negative (disconfirming) CF:

$$\begin{aligned}
\text{CF}_{\text{COMBINE}}(\text{CF}_p, \text{CF}_n) &= (\text{CF}_p + \text{CF}_n) / (1 - \min\{|\text{CF}_p|, |\text{CF}_n|\}) \\
&= (s_1 - s_3) / (1 - \min\{s_1, s_3\}) \\
&= (0.4 - 0.8) / (1 - 0.4) \\
&= -0.667.
\end{aligned}$$

Adapting this certainty factor to the language of the D-S theory, the result of the CF combining function is belief in {Pseu} and $\{\text{Pseu}\}^c$ to the degree 0 and 0.667, respectively. The larger disconfirming evidence of 0.8 completely negates the smaller confirming evidence of 0.4. The confirming evidence reduces the effect of the disconfirming from 0.8 to 0.667.

If one examines $\text{CF}_{\text{combine}}$ applied to combinations of confirming and disconfirming evidence as shown here, it is clear that it results in a CF whose sign is that of the CF with the greater magnitude. Thus, support for A and A^c is combined into reduced support for one or the other. In contrast, the D-S function results in reduced support for both A and A^c, a behavior that may more realistically reflect the competing effects of conflicting pieces of evidence.

The difference in the two approaches is most evident in the case of aggregation of two pieces of evidence, one confirming A to degree s and the other disconfirming A to the same degree. The CF function yields $\text{CF} = 0$ whereas

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[6] The CF combining function shown here has been used in MYCIN systems for several years but is slightly different from the formula described in the original CF model [15]. The revised empirically derived function prevents single pieces of positive or negative evidence from overwhelming the effect of several pieces of evidence in the opposite direction. The combining function remains unchanged from its original form, however, when applied to two pieces of evidence that are either both confirming or both disconfirming. See [3, Chap. 10] for a more detailed discussion of these points.

the D-S rule yields reduced but nonzero belief in each of A and A^c. We believe that the D-S rule's behavior in this case is preferable on the grounds that the notion of applying confirming and disconfirming evidence of the same weight should be different from that of having no evidence at all.

We now examine the effect on belief of combination of two pieces of evidence supporting mutually exclusive singleton hypotheses. The CF combining function results in no interaction between the beliefs in the two hypotheses and differs most significantly from the D-S rule in this case.

Theorem 3. *The rules involve different hypotheses in the same frame of discernment. For example, one rule confirms $\{Pseu\}$ to degree 0.4 (see m_1 in the examples from Categories 1 and 2) and the other disconfirms $\{Strep\}$ to degree 0.7. The application of the second rule corresponds to m_4, defined by $m_4(\{Strep\}^c) = 0.7$, $m_4(\Theta) = 0.3$. The combined effect on belief is given by $m_1 \oplus m_4$.*

m_1 $\diagdown$ m_4	$\{Strep\}^c\,(0.7)$	$\Theta(0.3)$
$\{Pseu\}\,(0.4)$ $\Theta\,(0.6)$	$\{Pseu\}\,(0.28)$ $\{Strep\}^c\,(0.42)$	$\{Pseu\}\,(0.12)$ $\Theta\,(0.18)$

In this case $\kappa = 0$ since the empty set does not occur in the tableau.

$$m_1 \oplus m_4\,(\{Pseu\}) = 0.28 + 0.12 = 0.40,$$
$$m_1 \oplus m_4\,(\{Strep\}^c) = 0.42,$$
$$m_1 \oplus m_4\,(\Theta) = 0.18,$$

$m_1 \oplus m_4$ is 0 for all other subsets of Θ.

$$Bel_1 \oplus Bel_4\,(\{Pseu\}) = 0.40,$$
$$\begin{aligned} Bel_1 \oplus Bel_4\,(\{Strep\}^c) &= m_1 \oplus m_4\,(\{Strep\}^c) + m_1 \oplus m_4\,(\{Pseu\}) \\ &= 0.42 + 0.40 \\ &= 0.82, \end{aligned}$$
$$Bel_1 \oplus Bel_4\,(\{Pseu\}^c) = Bel_1 \oplus Bel_4\,(\{Strep\}) = 0.$$

Before combination, the belief intervals for $\{Pseu\}$ and $\{Strep\}^c$ are $[0.4, 1]$ and $[0.7, 1]$, respectively. After combination, they are $[0.4, 1]$ and $[0.82, 1]$, respectively. Note that evidence confirming $\{Pseu\}$ has also confirmed $\{Strep\}^c$, a superset of $\{Pseu\}$, but that evidence confirming $\{Strep\}^c$ has had no effect on belief in $\{Pseu\}$, a subset of $\{Strep\}^c$. This kind of interaction among competing hypotheses is ignored by the CF model.

3.4 Evidence-combination Scheme

Although the calculations in Categories 1–3 in the previous section were straightforward, their simplicity is misleading. As the number of elements

in Θ increases, Barnett [2] has shown that direct application of the D-S theory, without attention to the order in which the *bpa*'s representing rules are combined, results in exponential increases in the time for computations. This is due to the need to enumerate all subsets or supersets of a given set. For settings in which it is possible to restrict the hypotheses of interest to singletons and their negations, Barnett has proposed a scheme for reducing the D-S computations to polynomial time by combining the functions in an order that simplifies the calculations. We outline this scheme as it could be adapted to reasoning system (such as MYCIN) in which evidence bears on mutually exclusive singleton hypotheses.

Step 1. For each triple (i.e., singleton hypothesis), combine all *bpa*'s representing rules confirming that value of the parameter. If s_1, s_2, ..., s_k represent different degrees of support derived from the triggering of k rules confirming a given singleton, then the combined support is $1 - (1 - s_1)(1 - s_2) \cdots (1 - s_k)$. (Refer to the example in Theorem 1 above for an illustration of this kind of combination. The formula shown here may be easily derived and is identical to the combining function used in the original CF model). Similarly, for each singleton, combine all *bpa*'s representing rules disconfirming that singleton. The same combining function is used for this calculation, and the numerical beliefs can simply be associated with the negation of the singleton hypotheses; it is not necessary to enumerate explicitly the elements in the set of size $n - 1$ (where n is the size of Θ) that corresponds to the complement of the singleton hypothesis in question. Thus, all evidence confirming a singleton is pooled and represented by a *bpa* and all evidence disconfirming the singleton (confirming the hypothesis corresponding to the set complement of the singleton) is pooled and represented by another *bpa*. We thus have $2n$ *bpa*'s, half of which assign belief to a singleton hypothesis and Θ (and which assign zero to all other hypotheses), the other half of which assign belief to the negation of a singleton hypothesis and Θ. Except for the notion of Θ, this step is identical to the original CF model's approach for gathering positive and negative evidence into the total confirming and disconfirming evidence respectively (MB and MD; see [15]).

Step 2. For each triple (singleton hypothesis), combine the two *bpa*'s computed in Step 1. Such a computation is a Theorem-2 combination and has been illustrated. Formulae that permit this calculation without the enumeration of any but the singleton subsets in 2^Θ are derived in [2] and described with examples in [9]. This step results in the definition of n *bpa*'s, one for each of the n singleton hypotheses. Each *bpa* that results assigns belief to a singleton hypothesis, its complement, and Θ while assigning zero to all other hypotheses.

Step 3. The final task is to blend all n *bpa*'s from Step 2 into a single belief function. This can be accomplished by combining the *bpa*'s derived in Step 2 in one computation, using formulae developed by Barnett to obtain the final belief function *Bel* [2]. Since these formulae allow computation of

both the net belief in a singleton A and in its negation A^c, the belief interval $[Bel(A),\ 1 - Bel(A^c)]$ for each singleton hypothesis can then be computed.

The details of Barnett's approach are described in [2]. In another publication, we have also provided the form of the required computation and have shown an example based on a small MYCIN rule set [9]. Since the new method proposed in the next section borrows only on Step 1 of the Barnett approach, we will not show the details of Steps 2 and 3 here.

4 The Dempster–Shafer Theory Applied to a Hierarchical Hypothesis Space

In a system in which all evidence either confirms or disconfirms singleton hypotheses, the combination of evidence via the D-S scheme with Barnett's formulae can be computationally simple as outlined in the previous section. As we have shown, a program such as MYCIN could be easily recast to use the D-S approach rather than the CF model.[7]

What attracted us to the D-S theory, however, and left us dissatisfied with the approach to singleton hypotheses proposed by Barnett, is the theory's potential for handling evidence bearing on categories of diseases as well as on specific disease entities. We are unaware of another model that suggests how evidence concerning hierarchically-related hypotheses might be combined coherently and consistently to allow inexact reasoning at whatever level of abstraction is appropriate for the evidence that has been gathered. The pure D-S model provides such a method for handling the aggregation of evidence gathered at varying levels of detail or specificity. Much of our frustration with the original MYCIN representation scheme and the CF model resulted from their inability to handle such hierarchical relationships cleanly. In recent years, a recurring theme in AI has been the explicit representation of hierarchic relationships among hypotheses (e.g., [8, 13]). Thus the D-S scheme might be especially suitable for handling uncertainty in such hierarchically organized networks. The problem, as we have emphasized, is the theory's computational complexity due to the potential need to enumerate all subsets in 2^Θ. Thus we have sought a technique that allows the model's use in a hierarchical hypothesis space while avoiding the exponential-time requirements that the theory

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[7] Additional conventions similar to those adopted in the CF model would be needed before the D-S approach could be used, however. For example, it would be necessary to adopt some mechanism for propagation of uncertainty in a rule-chaining environment. Barnett's suggestion [2] that MYCIN is ill-suited to such as implementation (due to its failure to satisfy the mutual exclusivity requirement) reflects a misunderstanding of the program's representation and control mechanisms. Multiple diseases are handled by instantiating each as a separate context (object); within a given context, the requirements of single-valued parameters (attributes assumed to take on precisely one value) maintain mutual exclusivity [3].

otherwise would entail. Since Barnett's approach is applicable only when the space is limited to singleton hypotheses and their negations, it will not serve our purposes.

To illustrate the need for such a capability, consider the way in which hierarchic relationships in the MYCIN domain were handled in that program. An example would be evidence suggesting that an organism was one of the Enterobacteriaceae (a family of gram-negative rods). The triple (hypothesis) for this conclusion was handled as (Organism Class Enterobacteriaceae), i.e., the frame of discernment (the Class parameter) was different from that normally used for concluding the identity of an organism (the Ident parameter). There was no way for the system to reach conclusions about both singleton hypotheses (e.g., Ident = E.coli) and supersets (e.g., Ident = Enterobacteriaceae) within the single Ident frame of discernment. Thus the Class parameter was introduced to handle the latter case. The relationship between Class Enterobacteriaceae and the individual organisms that make up that class was handled using rules in which evidence for Enterobacteriaceae was effectively transferred to Ident. This was accomplished by assigning as the values of the Ident parameter each of the bacteria on the list of gram negative organisms in that Class. The CF's assigned to the individual organism identities in this way were based more on guesswork than on solid data. The evidence really supported the higher-level concept, Enterobacteriaceae, and further breakdown may have been unrealistic. In actual practice, decisions about treatment are often made on the basis of high level categories rather than specific organism identities (e.g., "I'm quite sure that this is one of the enterics (i.e., the Enterobacteriaceae), and would therefore treat with an aminoglycoside and a cephalosporin (i.e., two types of antibiotic), but I have no idea which of the enteric organisms is causing the disease.").

Problems such as this would be better handled if experts could specify rules which refer to semantic concepts at whatever level in the domain hierarchy is most natural and appropriate. They should ideally not be limited to the most specific level—the singleton hypotheses in the frame of discernment—but should be free to use more unifying concepts. Because of the complexity in the D-S theory's approach to handling evidence, then, the challenge is to make these computations tractable, either by a modification of the theory or by restricting the evidence domain in a reasonable way. By taking the latter approach, we have developed an algorithm for the implementation of the theory which merges a strict application of the D-S combining function with a simplifying approximation.

4.1 Simplifying the Evidence Domain to a Tree Structure

The key assumption underlying our proposed approach is that the experts who participate in the construction of large knowledge bases can define a strict hierarchy of hypotheses about which the reasoning system will gather evidence. In D-S terms, we are suggesting that, for a given domain, only some

of the subsets in 2^Θ will be of semantic interest and that these can be selected to form a strict hierarchy. In medical diagnosis, for example, evidence often bears on certain disease categories as well as on specific disease entities. In the simplified case of cholestatic jaundice discussed earlier, for which $\Theta =$ {Hep, Cirr, Gall, Pan}, evidence available to the physician tends to support either intrahepatic cholestasis = {Hep, Cirr}, extrahepatic cholestasis = {Gall, Pan}, or the singleton hypotheses {Hep}, {Cirr}, {Gall}, and {Pan}. The other nodes of 2^Θ shown in Fig. 1 are not particulary meaningful notions in this context. The network of subsets in Fig. 1 could thus be pruned to that of Fig. 2, which summarizes the hierarchical relations of clinical interest. The hierarchy of Fig. 2 is a tree in the strict sense—each node below Θ has a unique parent. In the medical expert system known as MDX, the causes of jaundice have been usefully structured in precisely this way [4]. We believe, as do others [13], that such a structuring is characteristic of medical diagnostic tasks (as well as of many other problem-solving situations).

4.2 Evidence Combination Scheme for a Strict Hierarchy

We now propose a new three-step scheme for the implementation of the D-S theory in the situation in which the hypotheses of interest have been restricted by domain experts to subsets which form a strict hierarchy. It should be noted that, in general, the negations of hypotheses in the hierarchy (i.e., their set complements) will not be in the tree. For example, {Hep}^{c} = {Cirr, Gall, Pan} does not occur in the hierarchy of Fig. 2. Thus, as did Barnett in his Step 1, we propose an approach in which disconfirming evidence is handled computationally by associating it directly with the disconfirmed hypothesis rather than by converting it to be manipulated as confirming evidence regarding the complement of the disconfirmed hypothesis. The first two steps in our approach are a strict application of the D-S theory, in which simple formulae can be derived due to the tree structure of the hypotheses of interest. In the first step all confirmatory evidence is combined for each node in the tree, and the same is done for all disconfirmatory evidence. This step is similar to the first step in Barnett's approach (Sect. 3.4) except that the hypotheses are not restricted to singletons. In the second step all confirmatory evidence is combined for the entire tree. The third step is an approximation for combining
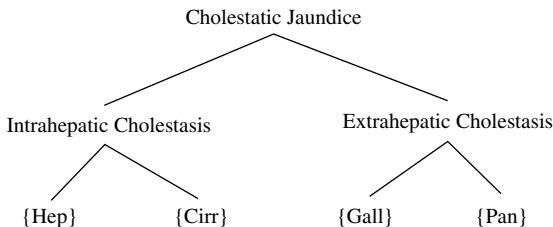

Fig. 2. The subsets of clinical interest in cholestatic jaundice

disconfirmatory evidence. Strict application of the D-S theory in this step may result in an exponential-time computation, whereas our approximation is computationally more efficient.

To illustrate these formulae, we use a slightly expanded version of the cholestatic-jaundice tree depicted in Fig. 2. Suppose we add to Θ a fifth cause of cholestatic jaundice, impaired liver function due to effects of oral contraceptives, denoted Orcon = {Orcon}. This addition will permit us to better demonstrate the properties of the technique we are proposing. Note that now Θ = cholestatic jaundice = {Hep, Cirr, Orcon, Gall, Pan} whereas intrahepatic cholestasis becomes the three-element subset {Hep, Cirr, Orcon} and has three direct descendents {Hep}, {Cirr}, and {Orcon}. This new tree is shown in Fig. 3 with only the first letter of each singleton hypothesis used, and commas and set brackets omitted for convenience of notation.

For the general case, we shall let T denote the set of all subsets (except for Θ itself) in the hierarchy of hypotheses that has been defined by the domain expert. Note that T is itself a subset of 2^{Θ}. However, it is convenient to think of T as simply the hypothesis tree without Θ. In our example, T is the set consisting of intrahepatic cholestasis, extrahepatic cholestasis, and the five single disease entities—i.e., {HCO, GP, H, C, O, G, P}. Let T' denote the set of all complements of subsets in T. T' is also a subset of 2^{Θ}, but the entities in T' will generally not be in T and hence are of interest only because they correspond to negations of pertinent hypotheses. In this example, T' is the set {HCO^c, GP^c, H^c, C^c, O^c, G^c, P^c}.

Step 1. Using the combining functions described in Step 1 of Barnett's evidence-combination scheme detailed in Sect. 3.4, for each subset X_i in T, combine all confirmatory evidence to obtain a *bpa*, m_{X_i}, and all disconfirmatory evidence to obtain another *bpa*, $m_{X_i^c}$.[8] Note that m_{X_i} can have a nonzero value on only X_i and Θ, $m_{X_i^c}$ on only X_i^c and Θ. Using our example, we would thus compute the following *bpa*'s: m_{HCO}, m_{GP}, m_{H}, m_{C}, m_{O}, m_{G}, m_{P},
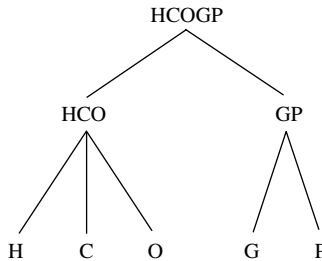

Fig. 3. The expanded tree of cholestatic jaundice

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[8] Note that we have introduced a variation on the notation used up to this point: m_i has denoted the *bpa* associated with the ith piece of evidence, whereas m_{X_i} denotes the *bpa* associated with the set X_i after all evidence confirming X_i has been combined.

m_{HCO^c}, m_{GP^c}, m_{H^c}, m_{C^c}, m_{O^c}, m_{G^c}, m_{P^c}. Thus, $m_{\text{HCO}}(\text{HCO})$ is the belief in intrahepatic cholestasis (i.e., HCO) after all evidence confirmatory of this disease category has been combined. The remaining belief, $1 - m_{\text{HCO}}(\text{HCO})$, is assigned to Θ. Similarly, $m_{\text{HCO}^c}(\text{HCO}^c)$ is the total belief against intrahepatic cholestasis and $1 - m_{\text{HCO}^c}(\text{HCO}^c)$ is assigned to Θ.

Our goal is to compute the single aggregate *bpa* that assigns net belief to all elements of T (by definition the only hypotheses of semantic interest for the domain) by blending in the disconfirming evidence associated with the sets in T'. This corresponds to the *bpa*

$$m_{Y_1} \oplus m_{Y_2} \oplus \cdots$$

where Y_i takes on the value of all subsets occurring in either T or T'. However, a strict application of the D-S theory in determining this *bpa* will assign nonzero values to many subsets that are in neither T nor T', precisely the event that we wish to avoid in order to prevent the enumeration of all sets in 2^Θ. The technique we propose combines in an organized fashion the *bpa*'s just computed in Step 1. Through a simple assumption defined below (see Step 3), we avoid the generation of new subsets.

We continue by observing that our aggregate final *bpa* can also be written as

$$m_T \oplus mT'$$

where

$$m_T = m_{x_1} \oplus m_{X_2} \oplus \cdots, X_i \in T$$

and

$$m_{T'} = m_{x_1^c} \oplus m_{X_2^c} \oplus \cdots, X_i^c \in T'.$$

The *bpa*, m_T, has nonzero values on only Θ or subsets in T, i.e., on $T \cup \Theta$, since the intersection of any two subsets in T is either the empty set or in T (the smallest of the two subsets). This computation is therefore performed as Step 2.

Step 2. Combine all confirmatory evidence by computing the aggregate *bpa*, m_T, of the *bpa*'s in Step 1 of the form m_{x_i}, where

$$m_T = m_{x_1} \oplus m_{x_2} \oplus \cdots, X_i \in T.$$

Note that m_T has nonzero value only on $T \cup \Theta$. In our example,

$$m_T = m_{\text{HCO}} \oplus m_{\text{GP}} \oplus m_{\text{H}} \oplus m_{\text{C}} \oplus m_{\text{O}} \oplus m_{\text{G}} \oplus m_{\text{P}}.$$

The quantity, $m_T(\text{HCO})$, is the belief in HCO (intrahepatic cholestasis) after combining all evidence confirmatory of this disease category with all evidence confirmatory of every other disease category or entity in the tree.

Note that the calculation in Step 2 does not include evidence *disconfirmatory* of HCO or the other hypotheses in T. That task is left to Step 3, i.e., the

remaining problem is to compute $m_T \oplus M_{T'}$. However, as mentioned earlier, if $m_{T'}$ is computed by a strict application of the D-S combining rule, it has nonzero value on many subsets that are in neither T nor T'. Even the aggregation of evidence disconfirmatory of a single subset in T (i.e., confirmatory of a single subset in T') with m_T leads to the generation of new subsets. For example, the combination of m_T with evidence disconfirmatory of hepatitis leads to a *bpa*, $m_T \oplus m_{H^c}$, which assigns belief to the diagnosis of CO, i.e., the set {Cirr, Orcon}.[9] This set is not in the tree of Fig. 3 because it was not originally defined to be of diagnostic interest. If this *bpa* is then combined with that representing evidence disconfirmatory of cirrhosis, belief is assigned to the diagnosis of HO = {Hep, Orcon}. This set also is not in T. As more *bpa*'s are aggregated via the D-S combination rule, more subsets are generated which are not in T and thus not of diagnostic interest. Hence, we make the approximation described in Step 3.

Step 3. Combine disconfirmatory evidence by step-wise combination of the $m_{X^c_i}$'s in the following way. Choose any set X^c_1 in T' and compute $m_T \ominus m_{X^c_1}$, which is an approximation to $m_T \oplus m_{X^c_1}$ with the property that $m_T \ominus m_{X^c_1}$ has nonzero value on only $T \cup \Theta$. Belief assigned to a subset A by $\oplus$ is instead assigned by $\ominus$ to the smallest superset of A in T if A itself is not in T. Now choose another set, X^c_2, in T', and compute $(m_T \ominus m_{X^c_1}) \ominus m_{X^c_2}$. Continue until all sets in T' have been chosen. The result is an aggregate *bpa* in which belief assigned to a set A in 2^{Θ} by the D-S function is sometimes assigned instead to an ancestor of A in $T \cup \Theta$. It may be shown (see Appendix A) that such an assignment is unique. Belief is thus displaced upward in the tree in order to avoid consideration of subsets not in T. Note that belief in A implies belief in B if B is a superset of A. The function, $\ominus$, is order-independent except in an easily identifiable case (see Appendix A).

To illustrate, belief assigned in the previous example to CO, a set not in the tree, is instead assigned to HCO, the smallest set in the tree containing it. Belief assigned to HO is also assigned to HCO. Note that disbelief in a singleton, which is represented as belief in its complement, is assigned by the approximation as belief in Θ (unless the complement happens to be in T).

As we have noted, the final *bpa* obtained by step-wise application of the function $\ominus$ in Step 3 differs from that obtained by the D-S function in that some belief assigned to a given subset by the latter is assigned to an ancestor of that subset by the former. Since belief in a subset of hypotheses implies belief in a superset of that subset, the upward displacement of belief in the hierarchy seems to be a reasonable exchange for the computational simplicity of our approximation method.

A final point is important to stress regarding the approach in Step 3. It should be clear that the scheme assigns *all* belief to subsets in T or to Θ. Thus, for A in *T, Bel(A)* can be computed by summing net belief in A with belief

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[9] Note that $m_T \oplus m_{H^c}$ assigns the quantity of belief, $m_T(\text{HCO})m_{H^c}(\text{H}^c)$ to CO = $\text{HCO} \cap \text{H}^c$.

assigned to all its descendents. However, it will not in general be possible to compute $Bel(A^c)$ since A^c will usually be in T' but not in T. Thus the notion of a belief interval, $[Bel(A), \ 1 - Bel(A^c)]$ is lost in the scheme we have proposed. Competing hypotheses would need to be compared based upon Bel alone without regard to the width of the plausibility interval (see Sect. 2.5).

In summary, the proposed evidence aggregation scheme is as follows.

Step 1. Calculate m_{X_i} for all X_i in T and $m_{X_i^c}$ for all X_i^c in T'.

Step 2. Calculate $m_T = m_{X_1} \oplus m_{X_2} \oplus \cdots$ for all X_i in T.

Step 3. Calculate $m_T \ominus m_{X_1^c}$, then $(m_T \ominus m_{X_1^c}) \ominus m_{X_2^c}$, etc. for all X_i^c in T'.

Recall that Step 1 is accomplished using the technique described in Sect. 3.4 and does not require the assumption of the tree structure of the domain or an approximation technique. Steps 2 and 3 do depend upon the assumption of the tree structure, however, and Step 3 requires the approximation outlined above. The formulae for the calculations in Steps 2 and 3 are given below, with their derivations provided in an Appendix A.

Step 2.

$$m_T(A) = \begin{cases} Km_A(A) \prod_{\substack{X \in T \\ X \not\supseteq A}} m_X(\Theta) & \text{if } A \in T, \\[2em] K \prod_{X \in T} m_X(\Theta) & \text{if } A = \Theta, \end{cases}$$

where $K = 1/(1 - \kappa)$ and

$$1 - \kappa = \sum_{A \in T} \left[m_A(A) \prod_{\substack{X \in T \\ X \not\supseteq A}} m_X(\Theta) \right].$$

Step 3. There are different formulae in Step 3 depending upon which of three relationships hold between X and A : $X \subseteq A, X \cap A = \emptyset$, or $X \supset A$, where X is a subset of $T \cup \Theta$ and A is a subset of T. In all cases, $K = 1/(1 - \kappa)$ where

$$\kappa = m_{A^c}(A^c) \sum_{\substack{X \in T \\ X \subseteq A}} m_T(X).$$

Case 1. $X \subseteq A$:

$$m_T \ominus m_{A^c}(X) = Km_T(X) m_{A^c}(\Theta).$$

Case 2. $X \cap A = \emptyset$ (i.e., $X \cap A^c = X$):

(i) If $X \cup A$ is a set in $T \cup \Theta$:

$$m_T \ominus m_{A^c}(X) = K[m_T(X) + m_T(X \cup A) m_{A^c}(A^c)].$$

(ii) If $X \cup A$ is not in $T \cup \Theta$:

$$m_T \ominus m_{A^c}(X) = Km_T(X).$$

Case 3. $X \supset A$:

(i) If $X \cap A^c$ is not a set in T:

$$m_T \ominus m_{A^c}(X) = Km_T(X).$$

(ii) If $X \cap A^c$ is in T:

$$m_T \ominus m_{A^c}(X) = Km_T(X) m_{A^c}(\Theta).$$

5 Conclusion

A major drawback for practical implementation of the Dempster–Shafer theory of evidence in reasoning systems has been its computational complexity (and resulting inefficiency). Based on the observation that evidence used in diagnostic reasoning involves abstract categories that can often be naturally represented in a strict hierarchical structure, we have designed a method for evidence aggregation based on the D-S theory. Using combinatorial analysis, a strict application of the theory, and an approximation, we have presented an approach which is computationally tractable.

Some observers may question the *value* of using the D-S scheme rather than the CF model or some other *ad hoc* method for handling uncertainty when dealing only with singleton hypotheses. Systems like MYCIN and INTERNIST-1 have demonstrated expert-level performance using their current techniques for inexact reasoning [12, 18]. We have previously suggested, in fact, that the details of a model of evidential reasoning in an AI system may be relatively unimportant since the careful semantic structuring of a domain's knowledge seems to blunt the sensitivity of its inferences to the values of the numbers used.[10] Some have even suggested that evidential reasoning can be handled without the use of a numerical model at all [5]. As was emphasized in Sect. 4, however, it is the D-S theory's techniques for managing reasoning about hypotheses in hierarchic abstraction spaces that we have found particularly appealing. The failure of previous models to deal coherently with these

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[10] See [3, Chap. 10] for a discussion of this point and an analysis of the sensitivity of MYCIN's conclusions to the CF values used in its rules. As is discussed there, MYCIN's performance can be shown to extremely insensitive to rather wide variations in the CF's assigned to its rules.

issues has led to unnatural knowledge representation schemes that require evidential associations among related concepts to be stated explicity rather than provided automatically by the hierarchic structure of pertinent domain concepts.

Directions for further work lie in the implementation and evaluation of our method in an actual reasoning system. Additional conventions will need to be defined before this can be done. For example, it is common for the evidence itself to be of an uncertain nature, and partially supported hypotheses in one frame of discernment may themselves be used as evidence to assign belief to hypotheses in another frame of discernment. This is a key feature of rule-chaining systems, for example, where belief in the premise conditions of rules may be less than certain. The *ad hoc* methods being used currently (e.g., the CF model's multiplicate convention [15]) may simply be borrowed for a D-S implementation. More interesting, perhaps, is the issue of how best to *use* the belief in the hypotheses after the proposed scheme has been applied. There is not likely to be a 'correct' approach to this problem because the nature of the actions based on evidence varies so greatly from one domain to another. Heuristics may be devised, however, for using thresholding or relative belief measures to determine what level of abstraction in the hypothesis hierarchy is most appropriately selected as the basis for a final conclusion or recommendation from an advice system.

The techniques described here will be neither necessary nor adequate for all expert system application domains. Some tasks are well managed by purely categorical inference techniques, and others do not lend themselves to hierarchical domain structuring and the evidence gathering model of problem solving. However, for diagnostic or classification tasks in settings where the hypothesis space is well suited to assumptions of mutual exclusivity and hierarchical organization, we believe that our adaptation of the Dempster–Shafer theory holds great appeal as a computationally tractable and coherent belief model.

Appendix A

We present here the details of Steps 2 and 3 in the proposed evidence combination scheme outlined in Sect. 4.

A Step 2: Aggregation of Confirmatory Evidence

The *bpa* m_T is the aggregate of all *bpa*'s of the form, m_X, where X is a subset in T. Each m_X has been obtained by combining all confirmatory evidence for X. For the following discussion we shall use A to refer to an arbitrary subset in $T \cup \Theta$. We now derive formulae for m_T by first computing the normalization constant, $K = 1/(1 - \kappa)$, and then $m_T(A)$ for any subset A in $T \cup \Theta$.

A.1 The Normalization Constant of m_T

Recall that $1 - \kappa$ is the sum of all beliefs not attributed to the empty set. Thus, $1 - \kappa$ is

$$\sum \prod_{X \in T} m_X(Y_X),$$

where Y_X is either X (a subset in T) or Θ and the Y_X's intersect to give a non-empty subset. For example, in the cholestatic jaundice hierarchy of Fig. 3, two of the summands in $1 - \kappa$ would be:

$$m_H(H) m_C(\Theta) \, m_O(\Theta) \, m_G(\Theta) \, m_p(\Theta) \, m_{GP}(\Theta) \, m_{HCO}(HCO),$$
$$m_H(H) m_C(\Theta) \, m_O(\Theta) \, m_G(\Theta) \, m_p(\Theta) \, m_{GP}(\Theta) \, m_{HCO}(\Theta).$$

Note that once we choose $Y_A = A$ for a specific A, then, in order to avoid the empty set as the final intersection, we must choose all other $Y_X = \Theta$ except for descendents (subsets) or ancestors (supersets) of A. In the above example, once we chose $Y_H = H$, we had to choose $Y_X = \Theta$ for all other X except for $X = HCO$, the one ancestor of H in T. For Y_{HCO}, we could choose Y_{HCO} as either HCO or Θ. Thus, we claim that

$$1 - \kappa = \sum_{A \in T} \left[m_A(A) \prod_{\substack{X \in T \\ X \not\supseteq A}} m_X(\Theta) \prod_{\substack{X \in T \\ X \supset A}} [m_X(X) + m_X(\Theta)] \right].$$

Since m_X has nonzero value on only X and Θ, $m_X(X) + m_X(\Theta) = 1$ for all X in T. Thus,

$$\prod_{\substack{X \in T \\ X \supset A}} [m_X(X) + m_X(\Theta)] = 1$$

and the above simplifies to

$$1 - \kappa = \sum_{A \in T} \left[m_A(A) \prod_{\substack{X \in T \\ X \not\supseteq A}} m_X(\Theta) \right].$$

The two products given above for the cholestatic jaundice example would be represented in this expression by the summand in the expression for $1 - \kappa$ formed by choosing $A = H$. Because $m_{HCO}(HCO) + m_{HCO}(\Theta) = 1$, note that these two summands add to $m_H(H) m_C(\Theta) m_O(\Theta) m_G(\Theta) m_P(\Theta) m_{GP}(\Theta)$.

A.2 Computation of $m_T(A)$

In order to derive a formula for $m_T(A)$, where A is any subset in $T \cup \Theta$, we need to enumerate all products of the form

$$\prod_{X \in T} m_X(Y_X),$$

where Y_X is either X (a subset in T) or Θ and the intersection of the Y_X's is A. For $A = \Theta$, we must choose $Y_X = \Theta$ for each X in T. Thus,

$$m_T(\Theta) = K \prod_{X \in T} m_X(\Theta).$$

For every A in T, we must choose $Y_A = A$ for the factor $m_A(Y_A)$ since $Y_A = \Theta$ will in general make it impossible to achieve a final intersection of A due to the tree structure of the subsets. If X is not an ancestor of A, then we must choose $Y_X = \Theta$ since $Y_X = X$ will yield an empty intersection for some subset of A. If X is an ancestor of A, then both $Y_X = X$ and $Y_X = \Theta$ will yield A as the intersection. Thus, we obtain

$$m_T(A) = K m_A(A) \prod_{\substack{X \in T \\ X \not\supseteq A}} m_x(\Theta) \prod_{\substack{X \in T \\ X \supset A}} [m_X(X) + m_X(\Theta)].$$

Once again, the indicated sum, and hence the last product, is 1 and the above simplifies to

$$m_T(A) = K m_A(A) \prod_{\substack{X \in T \\ X \not\supseteq A}} m_X(\Theta).$$

For example, in our model of cholestatic jaundice from Fig. 3, the effect of all confirmatory evidence on belief precisely in hepatitis is given by

$$m_T(\mathrm{H}) = K m_{\mathrm{H}}(\mathrm{H}) m_{\mathrm{C}}(\Theta) m_{\mathrm{O}}(\Theta) m_{\mathrm{G}}(\Theta) m_{\mathrm{P}}(\Theta) m_{\mathrm{GP}}(\Theta).$$

The effect on belief in intrahepatic cholestasis (i.e., HCO) is given by

$$m_T(\mathrm{HCO}) = K m_{\mathrm{HCO}}(\mathrm{HCO}) m_{\mathrm{H}}(\Theta) m_{\mathrm{C}}(\Theta) m_{\mathrm{O}}(\Theta) m_{\mathrm{G}}(\Theta) m_{\mathrm{P}}(\Theta) \\ m_{\mathrm{GP}}(\Theta).$$

B Step 3: Aggregation of Disconfirmatory Evidence

As mentioned in Sect. 4, it is in this step that we first depart from a strict application of the D-S combining function in order to avoid the assignment of belief to subsets which are neither in T nor T'. Our solution to this difficulty is an approximation, $m_T \ominus m_{A^c}$, which assigns all belief to subsets in $T \cup \Theta$; i.e., the subsets on which m_T may have nonzero value. For example, in the hierarchy of Fig. 3, belief that would be assigned to CO is instead assigned to its smallest ancestor in T, HCO. This is a justifiable assignment because:

– the subset CO is, by the domain expert's definition of T, not of diagnostic interest and so should not be assigned belief;
– evidence confirming a subset also logically supports supersets of that subset;
– there is a unique smallest superset due to the strict tree structure of the hierarchy defined by the subsets in T, i.e., each subset in T has precisely one parent in T, except for those at the top of the hierarchy whose parent is Θ.

Thus, $m_T \ominus m_{A^c}$ assigns $m_T(X)m_{A^c}(A^c)$ to $X \cap A^c$ if $X \cap A^c$ lies in $T \cup \Theta$ and to X (which can be shown to be the unique smallest superset in $T \cup \Theta$ containing $X \cap A^c$) if not. We now derive formulae for $m_T \ominus m_{A^c}$.

B.1 Computation of Normalization Constant for the Modified Combining Function

This time we consider κ, the sum of beliefs assigned to $\emptyset$, instead of $1 - \kappa$ as we did in Step 2. Thus, we want a simplified expression for

$$\kappa = \sum_{X \in T} m_T(X)\, m_{A^c}(Y_{A^c}),$$

where $Y_{A^c} = A^c$ or Θ and $X \cap Y_{A^c} = \emptyset$. Clearly, X and Y_{A^c} are not disjoint if $Y_{A^c} = \Theta$. If $Y_{A^c} = A^c$, then we must choose $X = A$ or X a subset of A to yield $X \cap Y_{A^c} = \emptyset$. Thus,

$$\kappa = m_{A^c}(A^c) \sum_{\substack{X \in T \\ X \subseteq A}} m_T(X).$$

B.2 Formulae for the Modified Combining Function

We derive formulae for $m_T \ominus m_{A^c}(X)$ where X lies in $T \cup \Theta$ and therefore falls into one of three cases. We are looking in each case for all sets in $T \cup \Theta$ which intersect with either A^c or Θ to give X. For purposes of illustration, consider the hypothesis tree of Fig. 3 and the calculations necessary for combining evidence disconfirmatory of pancreatic cancer ($A = \mathrm{P}$).

Case 1. $X \subseteq A$. There is no subset in $T \cup \Theta$ that will intersect with A^c to give X so the only possibility is to choose X and Θ to yield $X \cap \Theta = X$. Thus,

$$m_T \ominus m_{A^c}(X) = K m_T(X)\, m_{A^c}(\Theta).$$

In our example with $A = \mathrm{P}$, the only set X in this case is $X = A = \mathrm{P}$. Thus,

$$m_T \ominus m_{\mathrm{p}^c}(\mathrm{P}) = K m_T(\mathrm{P})\, m_{\mathrm{p}^c}(\Theta).$$

Case 2. $X \cap A = \emptyset$ (i.e., $X \cap A^{c} = X$). Note that we may choose either X, A^{c} or X, Θ as pairs yielding an intersection equal to X. Two subcases should be distinguished: that in which $X \cup A$ is in $T \cup \Theta$ and that in which $X \cup A$ is not. For if $X \cup A$ is in $T \cup \Theta$, then we may also choose the pair $X \cup A, A^{c}$ to yield X as the intersection. Thus, in the first subcase:

$$m_T \ominus m_{A^c} (X) = K[m_T (X) m_{A^c} (A^c) + m_T (X) m_{A^c} (\Theta)$$
$$+ m_T (X \cup A) m_{A^c} (A^c)].$$

This expression simplifies to

$$m_T \ominus m_{A^c} (X) = K [m_T (X) + m_T (X \cup A) m_{A^c} (A^c)],$$

since $m_{A^c}(A^c) + m_{A^c}(\Theta) = 1$.

In our example with $A = \mathrm{P}$, the set G falls into this subcase and

$$m_T \ominus m_{\mathrm{p}^c}(\mathrm{G}) = K[m_T (\mathrm{G}) + m_T (\mathrm{GP}) m_{\mathrm{p}^c} (\mathrm{P}^c)].$$

The second subcase applies for all X in T such that X, A^c and X, Θ are the only two pairs yielding an intersection equal to X:

$$m_T \ominus m_{A^c} (X) = K[K m_T (X) m_{A^c} (A^c) + m_T (X) m_{A^c} (\Theta)],$$

which simplifies to
$$m_T \ominus m_{A^c} (X) = K m_T (X),$$

since $m_{A^c}(A^c) + m_{A^c}(\Theta) = 1$.

In our example with $A = \mathrm{P}$, the subsets HCO, H, C, and O fall in this subcase and thus

$$m_T \ominus m_{\mathrm{p}^c} (\mathrm{HCO}) = K m_T (\mathrm{HCO}),$$
$$m_T \ominus m_{\mathrm{p}^c} (\mathrm{H}) = K m_T (\mathrm{H}),$$
$$m_T \ominus m_{\mathrm{p}^c} (\mathrm{C}) = K m_T (\mathrm{C}),$$
$$m_T \ominus m_{\mathrm{p}^c} (\mathrm{O}) = K m_T (\mathrm{O}).$$

Case 3. $X \supset A$: In this case, the only pair yielding an intersection of X is X, Θ. However, consider the pair X, A^c whose intersection may or may not lie in T. If $X \cap A^c$ does not lie in T, it may be shown that X is the smallest superset of $X \cap A^c$ containing $X \cap A^c$ and we assign $m_T(X)m_{A^c}(A^c)$ to X. Then,

$$m_T \ominus m_{A^c}(X) = K[m_T(X)m_{A^c}(A^c) + m_T(X)m_{A^c}(\Theta)] = K m_T(X).$$

In our example, $m_T \ominus m_{\mathrm{Pc}}(\Theta) = K m_{\mathrm{T}}(\Theta)$, since $m_T(\Theta)m_{\mathrm{p}^c}(\mathrm{P}^c)$ is assigned to Θ, the smallest superset of P^c in $T \cup \Theta$.

If $X \cap A^c$ does lie in T, then $m_T(X)m_{A^c}(A^c)$ was assigned to $X \cap A^c$ in Case 2. Clearly, if $X \cap A^c$ is a subset in T, $X \cap A^c$ falls into Case 2 since $(X \cap A^c) \cap A = \emptyset$. Thus, for $X \supset A$ and $X \cap A^c \in T$:

$$m_T \ominus m_{A^c}(X) = Km_T(X)\,m_{A^c}(\Theta).$$

In our example, $m_T \ominus m_{\mathrm{P^c}}(\mathrm{GP}) = Km_T(\mathrm{GP})m_{\mathrm{P^c}}(\Theta)$.

B.3 Optimal Ordering of Evidence Aggregation

It can be shown that the function $\ominus$ is order-independent except in the case of evidence involving a subset A where both A and its parent have exactly one sibling. In the hierarchy shown in Fig. 3, for example, the configuration of concern occurs when A is taken to be either G or P. In this situation, evidence involving the higher level subset GP should be combined before that involving G or P. A small portion of the belief that would be assigned to G or P by the D-S function is correctly assigned to G or P if disconfirming evidence $m_{X_i^c}$ is aggregated first with the higher level subset and then with G and P. However, it is assigned to GP, the parent of G and P, if the disconfirming evidence is aggregated with the lower level subsets first.

Thus, a better approximation to the D-S function is obtained depending on the order for aggregation chosen in Step 3. However, this difference is insignificant in that the amount of belief involved is small and more importantly, it is only displaced upward by one level from a subset to its parent. Such upward displacement is a common result of the approximation function anyway. Combining evidence in a breadth-first fashion, from higher to lower levels, will result in an optimal approximation.

Acknowledgment

References

1. Adams, J. B., A probability model of medical reasoning and the MYCIN model, *Math. Biosci.* **32** (1976) 177–186.
2. Barnett, J. A., Computational methods for a mathematical theory of evidence, in: *Proceedings Seventh International Joint Conference on Artificial Intelligence*, Vancouver, BC (1981) 868–875.

3. Buchanan, B. G. and Shortliffe, E. H., *Rule-Based Expert Systems: The MYCIN Experiments of the Stanford Heuristic Programming Project* (Addison-Wesley, Reading, MA, 1984).

4. Chandrasekaran, B., Gomez, F., Mittal, S. and Smith, M., An approach to medical diagnosis based on conceptual schemes, in: *Proceedings Sixth International Joint Conference on Artificial Intelligence*, Tokyo, Japan (1979) 134–142.

5. Cohen, P. R., *Heuristic Reasoning about Uncertainty: An Artificial Intelligence Approach* (Pitman, London, 1984).

6. Friedman, L., Extended plausible inference, in: *Proceedings Seventh International Joint Conference on Artificial Intelligence*, Vancouver, BC (1981) 487–495.

7. Garvey, T. D., Lowrence, J. D. and Fischler, M. A., An inference technique for integrating knowledge from disparate sources, in: *Proceedings Seventh International Joint Conference on Artificial Intelligence*, Vancouver, BC (1979) 319–325.

8. Gomez, F. and Chandrasekaran, B., Knowledge organization and distribution for medical diagnosis, in: W. J. Clancey and E. H. Shortliffe (Eds.), *Readings in Medical Artificial Intelligence: The First Decade* (Addison-Wesley, Reading, MA, 1984) 320–338.

9. Gordon, J. and Shortliffe, E. H., The Dempster–Shafer theory of evidence, in: B. G. Buchanan and E. H. Shortliffe (Eds.), *Rule-Based Expert Systems: The MYCIN Experiments of the Stanford Heuristic Programming Project* (Addison-Wesley, Reading, MA, 1984) 272–292.

10. Gouvernet, J., Ayme, S., Sanchez, E., Mattei, J. F. and Giraud, F., Diagnosis assistance in medical genetics based on belief functions and a tree structured thesaurus: a conversational mode realization, in: *Proceedings of MEDINFO 80*, Tokyo, Japan (1980) 798.

11. Gouvernet, J., Apport des methods de classification en genetique medicale, Thesis, University of Marseilles, 1979.

12. Miller, R. A., Pople, H. E. and Myers, J. D., INTERNIST-1: An experimental computer-based diagnostic consultant for general internal medicine, *New England J. Medicine* **307**(8) (1982) 468–476.

13. Pople, H. E., Heuristic methods for imposing structure on ill-structed problems: the structuring of medical diagnostics, in: P. Szolovits (Ed.), *Artificial Intelligence in Medicine* (Westview Press, Boulder, CO, 1982) 119–190.

14. Shafer, G., *A Mathematical Theory of Evidence* (Princeton University Press, Princeton, NJ, 1976).

15. Shortliffe, E. H. and Buchanan, B. G., A model of inexact reasoning in medicine, *Math. Biosci.* **23** (1975) 351–379.

16. Strat, T. M., Continuous belief functions for evidential reasoning, in: *Proceedings Fourth National Conference on Artificial Intelligence*, Austin, TX (1984) 308–313.

17. Szolovits, P. and Pauker, S., Categorical and probabilistic reasoning in medical diagnosis, *Artificial Intelligence* **11** (1978) 115–44.

18. Yu, V. L., Fagan, L. M., Wraith, S. M., Clancey, W. J., Scott, A. C., Hannigan, J. F., Blum, R. L., Buchanan, B. G. and Cohen, S. N., Antimicrobial selection by a computer: a blinded evaluation by infectious disease experts, *J. Amer. Med. Assoc.* **242** (1979) 1279–1282.

19. Zadeh, L. A., A mathematical theory of evidence (book review), *AI Magazine* **5**(3) (1984) 81–83.